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Soil Lead - Blood Lead Relationship in a Former Lead Mining Town

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Abstract

The impact of exterior surface dust lead and soil lead on children's blood lead has been estimated on two distinctly different cohorts of children. The two studies employed the same environmental and biological sample collection and analytical procedures, as well as data analysis procedures. Both studies revealed an indirect association between exterior surface dust lead and blood lead. This association was mediated by lead contamination of interior house dust and subsequent contamination of children's hands.

Introduction

Many studies have examined the association between soil and/or dust lead and children's blood lead. Success in demonstrating such an association is influenced by the multiplicity of sources of lead in a child's environment and the complexities of children's interactions with their environment. There is a general consensus that soil lead can increase blood lead in children. However, the magnitude of the impact and the conditions which modify the impact remain unclear. For example, the Centers for Disease Control (Preventing Lead Poisoning in Young Children, 1985) concludes that "lead in soil and dust appears to be responsible for blood lead levels in children increasing above background levels when the concentration in soil or dust exceeds 500-1,000 $\mu\text{g/g}$ ". Others (Duggan and Inskip, 1985; Chaney and Mielke, 1986) have reviewed the literature and concluded that the average impact is about 5 $\mu\text{g/dL}$ lead in blood/1,000 $\mu\text{g/g}$ lead in soil. They also noted the wide range of such estimates (1.5 to 9.0 $\mu\text{g/dL}$ per 1,000 $\mu\text{g/g}$). After a more detailed evaluation of a subset of these same studies, the Environmental Protection Agency (Air Quality Criteria for Lead, 1986) concluded that the effect size is about 2 $\mu\text{g/dL}$ per 1,000 $\mu\text{g/g}$ lead in soil. Most recently, the Committee on Environmental Hazards of the American Academy of Pediatrics (Statement on Childhood Lead Poisoning, 1981) stated that "Each increase of 100 $\mu\text{g/g}$ in the lead content of surface soil above a level of 500 $\mu\text{g/g}$ is associated with a mean increase

in children's whole blood lead levels of 1 to 2 $\mu\text{g/dL}$ ". This latter estimate is an order of magnitude higher than previously cited estimates. Many factors contributing to variation in the size of the soil lead-blood lead association have generally been ignored in arriving at these estimates. These include chemical speciation, particle size, environmental accessibility, bioavailability, behavioral differences in how children interact with their environment and their existing body lead burden.

For the past eight years, we have been investigating the role of social, biological and behavioral factors in determining the impact of environmental lead on blood lead levels in a cohort of inner city children (Bornschein *et al.*, 1985). As a result of these studies we have derived a model of childhood lead exposure which implicates an indirect path from exterior surface dust lead to interior house dust lead to lead on children's hands, ultimately resulting in a rise in children's blood lead levels (Bornschein *et al.*, 1985; Bornschein *et al.*, 1986).

In the autumn of 1986, we had an opportunity to confirm the validity of this model in a distinctly different cohort of children. This new cohort of children was largely middle class, residing in the small (population about 1,200) resort community of Telluride, Colorado. The town had previously been the site of an extensive lead mining and milling operation. At the time of closure of the mine in 1977, a blood lead survey was conducted. The average blood lead of 68 children, excluding children of miners or mill workers, was reported to be 17.5 $\mu\text{g/dL}$ (Telluride Survey: 1977). Four children of mine or ore mill workers had blood lead values greater than 30 $\mu\text{g/dL}$ and were presumably exposed to lead brought home on the clothing of the workers (*cf.* Rice *et al.*, 1978). Following closure of the mine, concern was voiced that the mill wastes or tailings, which covered a 60 acre area immediately east of the town, could be a continuing source of lead exposure for the town and its inhabitants. In 1986, a soil grid survey of the town revealed that 30% of the 126 soil cores contained lead at concentrations greater than 500 $\mu\text{g/g}$, while 17% were greater than 1,000 $\mu\text{g/g}$. Analysis of the lead content of the tailings revealed the average concentration to be 1,700 $\mu\text{g/g}$, with a range from 1,300 to 5,100 $\mu\text{g/g}$.

Given the previously cited associations between soil lead and blood lead in children, a decision was made to conduct a second blood lead survey in Telluride. Based on our experience with the Cincinnati cohort, we also decided to include measurements of lead in the children's immediate residential environment as well as public areas, playgrounds and schools frequented by the children.

Experimental Materials and Methods

Subjects

The survey consisted of 258 individuals. Ninety-four (36.4%) were children less than or equal to 72 months of age. An estimated 81% of all children, less than or equal to 72 months of age and residing in town were screened. Non-participants did not differ statistically from study participants with respect to important covariates and confounders. Other groups examined

included older children and young adults, pregnant women and occupationally exposed adults such as former mine employees, house painters, *etc.*

Blood lead determinations

Whole blood samples were collected by venipuncture using a 23 gauge butterfly needle. Blood samples were analyzed for lead using an ESA Model 3010A anodic stripping voltammeter. Details of our laboratory methods and quality control and quality assurance program have been provided elsewhere (Roda *et al.*, 1988). Approximately 10% of the samples were also analyzed in duplicate by Environmental Science Associates. Agreement between laboratories was high ($r = 0.97$).

Environmental Lead Determinations

Environmental samples were collected from 45 residences, five day care centres or schools, several babysitters' homes and all major parks and playgrounds in town. The types of samples collected included interior and/or exterior paint (PbP) samples, *in situ* x-ray fluorescence determinations of interior (XRF-INT) and exterior (XRF-EXT) paint lead content, two composite dust samples from the interior windowsills (PbD-Window) and bedroom, living room and kitchen floors (PbD-Floor), composite 2.5 cm deep, soil core (PbSC) samples from grassy areas around the perimeter of the house and along the street or sidewalk and soil surface scrapings (PbSS) from exposed soil in play areas, paths through yards or playgrounds and from paved areas immediately outside the house entry.

Water samples (PbW) were collected from the kitchen tap (one litre grab samples) and from public buildings. Handwipes (PbH) were obtained from each child at their residence to estimate the amount of lead adhering to their hands. Sample collection and analysis procedures have been described previously (Que Hee *et al.*, 1985; Bornschein *et al.*, 1986).

In addition, the location of each dwelling and public building was noted with respect to the previous soil lead grid survey, proximity to the nearest tailings pond (Dist. 1) and proximity to the old railway right of way (Dist. 2) which originated at the mill on the east side of town and ran parallel to the San Miguel river on the southern boundary of the town. This latter right-of-way coincided with the region of highest soil lead concentrations obtained in the soil lead grid survey.

Data Analyses

Environmental lead measures and blood lead measures were subjected to natural log transformation in order to normalize their distributions. Descriptive statistics are presented as geometric means and geometric standard deviations unless otherwise noted. Pearson "r" correlations were computed using these log transformed data. A descriptive model of presumed causal influences was derived with the aid of structural equations analyses. This technique provides estimates of the impact of direct and indirect influences on an outcome variable such as PbB (Bentler, 1980;

Bornschein, *et al.*, 1985; Bornschein *et al.*, 1986). The SAS SYSREG computer program (SAS, 1982) was used to estimate the structural parameters.

Results and Discussion

A brief summary of the results of the blood lead survey appears in Table 1. None of the 258 individuals exhibited evidence of excessive lead exposure as defined by the Centers for Disease Control (Preventing Lead Poisoning

Table 1. Telluride blood lead survey results.

Group	N	Average*	Minimum	Maximum
Children \leq 72 months	94	6.1	2.0	19.0
Adults without occupational exposure	117	5.6	2.0	13.5
Occupationally exposed adults	8	15.1	4.6	24.4
Pregnant women	12	3.5	1.9	6.2

*Arithmetic average in $\mu\text{g/dL}$.

**Past or present mine employees, house painters, etc.

in Young Children, 1985). These levels are likely to be representative of blood lead levels (PbB) obtained under normal rural conditions. Children living nearest to the tailings did not have blood leads higher than those living farther from the tailings (PbB = 6.7; $n = 6$ and PbB = 6.8; $n = 54$ respectively). The arithmetic average blood lead levels of those living in town (PbB = 6.8; $n = 54$) were identical to that seen in children residing out of town (PbB = 6.8; $n = 34$). As expected, past or present mine/mill employees had higher PbB levels than those without such exposure.

Table 2 provides descriptive statistics for the subset of 45 children for whom complete environmental, social and blood lead data were available. Contrary to the popular perception that lead based paint is found only in older urban inner cities in the East, we found that 75% of the residences surveyed had at least one surface that tested positive ($\text{XRF} \geq 0.7 \mu\text{g/cm}^2$) for lead based paint. Furthermore, 40% of the residences had high ($\text{XRF} \geq 6.0$) levels of lead in paint. The average drinking water lead content ($1.1 \mu\text{g/kg}$; range = 0.4 to $11.0 \mu\text{g/kg}$) was well below the US Public Health Service Drinking Water Standard of $50 \mu\text{g/kg}$. Soil lead and PbSS immediately around the residence as well as PbD on floors and windowsills were low and comparable to that seen in lead abated housing in inner-city Cincinnati (Clark *et al.*, 1985). In a few cases, extremely high dust lead levels were found in window-wells. This was most likely due to the presence of high lead content paint chips in the dust sample, since these same windows yielded high XRF readings. Hand wipe lead levels were also low and consistent with the low dust lead and blood lead values. Data in this table can be contrasted with that reported by Clark *et al.* (1985, 1988).

Table 2. Telluride survey descriptive statistics ($N = 45$).

Variable	Mean	S.D.	Minimum	Maximum
XRF-EXT (mg Pb/cm^2)	1.9	3.1	0.4	18.4
XRF-INT (mg Pb/cm^2)	2.6	4.1	0.2	25.3
PbSS ($\mu\text{g/g}$)	178	2.5	16	1,895
PbSC ($\mu\text{g/g}$)	145	3.2	17	804
PbD Floor ($\mu\text{g/g}$)	281	1.9	86	3,165
PbD Windowsill ($\mu\text{g/g}$)	567	5.0	42	147,267
AGE (years)	3.3	1.8	0.3	8.2
PbH (μg)	4.5	2.3	<1	50
PbB ($\mu\text{g/dL}$)	6.1	1.7	<2	19

PbH = hand Pb in μg for 2 hands

PbD = floor dust Pb in $\mu\text{g/g}$

XRF = maximum exterior XRF/house in mg/cm^2

PbSS = median surface scraping Pb/house in $\mu\text{g/g}$

PbSC = median 1 inch soil core Pb in $\mu\text{g/g}$

DIST. 1 = distance of house from tailings pond in feet

DIST. 2 = distance of house from railway and creek in feet

Table 3. Telluride survey correlation matrix.

	PbB	PbH	PbD	XRF	PbSS	PbSC
PbH	-0.30					
PbD	0.39	0.34				
XRF	NS	NS	NS			
PbSS	NS	NS	0.51	0.40		
PbSC	NS	NS	0.46	0.49	0.67	
DIST. 1	NS	NS	NS	NS	NS	0.43
DIST. 2	NS	NS	-0.38	NS	-0.35	NS

PbH = hand Pb in μg for 2 hands.

PbD = floor dust Pb in $\mu\text{g/g}$.

XRF = maximum exterior XRF/house in mg/cm^2

PbSS = median surface scraping Pb/house in $\mu\text{g/g}$

PbSC = median 1 inch soil core Pb in $\mu\text{g/g}$.

DIST. 1 = distance of house from tailings pond in feet

DIST. 2 = distance of house from railway and creek in feet

NS = $p > 0.05$

Correlations among biological and environmental measures of lead are shown in Table 3. Although lead concentrations were low, statistically significant ($p \leq 0.05$) correlations were observed. The negative simple correlation between PbH and PbB is not in the predicated direction. As will be shown later, child age is an important modifier of the relationship between PbH and PbB. The other somewhat anomalous association is the positive correlation between PbSC and Dist. 1, *i.e.* the further the residence was located from the tailings, the higher the concentration of lead in soil cores taken from around the residence. This is probably a reflection of lead's distribution in the town as a function of railway activity and flooding of the San Miguel River, causing tailings to be distributed along its banks, rather than representing a simple linear relationship of Pb in soil cores as a function of distance from the tailings. No simple, direct

Table 4. Correlations among soil, dust and hand lead.

		PbSS ($\mu\text{g/g}$)	PbH (μg)
PbD Floor	$\mu\text{g/g}$	0.51	0.34
	mg/m^2	0.52	0.34
PbD Window	$\mu\text{g/g}$	0.36	0.21
	mg/m^2	0.38	0.20

association was found to be significant between PbSS or PbSC and PbH or PbB.

Interior dust collection methods permitted lead to be expressed as amount of lead per unit weight of dust ($\mu\text{g/g}$) or amount per unit area ($\mu\text{g/m}^2$). Table 4 shows the correlation of these two units of measurement with exterior PbSS and children's PbH. Since the loading measure ($\mu\text{g/m}^2$) did not appear to offer any distinct advantage over the concentration measure ($\mu\text{g/g}$), and since the latter corresponds to the units used for exterior PbSS and PbSC $\mu\text{g/g}$ Pb in dust was selected for use in further modelling. Note also that PbD-Floor is more strongly correlated with PbSS and PbH than is PbD-Window. This is consistent with hypothesis that the exterior surface dust lead is tracked into the home and that young children come in contact with it on the floor, thereby contaminating their hands.

The results of a structural equations analysis of this data is summarized in Figure 1. The solid lines indicate that a statistically significant, covariate adjusted association exists between connected variables. The arrows indicate the presumed direction of causal influence. The numbers next to the arrows are estimates of the structural equations parameters. The dashed lines indicate that a significant association exists, but that no causal direction is assumed. The absence of a line connecting any two variables

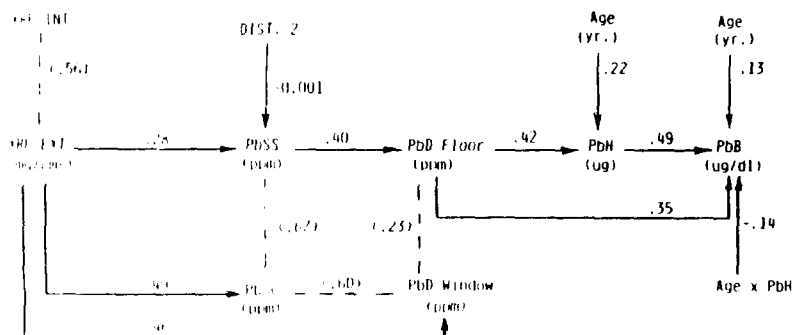


Figure 1. Final structural equations model showing the direct and indirect influences of environmental lead on blood lead. Arrows indicate the presumed direction of causal influences. Numbers next to the arrows are parameter estimates. Dashed lines link correlated variables between which no causal direction is assumed.

indicates that no significant association was found since all possible pathways were tested via a backward elimination strategy. The complete structural equation is presented below as a series of simultaneous regression equations.

Structural Equations

$$\begin{aligned} \text{Ln(PbB)} = & 0.545 + 0.494 \text{ Ln(PbH)} + 0.128 (\text{Age}) \\ & - 0.140 (\text{LnPbH} \times \text{Age}) + 0.347 \text{ Ln(PbD)} \end{aligned} \quad \begin{matrix} R^2 \\ 0.44 \end{matrix}$$

where

$$\text{Ln(PbH)} = -1.582 + 0.218 (\text{Age}) + 0.420 \text{ Ln(PbD)} \quad 0.32$$

and

$$\text{Ln(PbD)} = 3.573 + 0.400 \text{ Ln(PbSS)} \quad 0.45$$

and

$$\text{Ln(PbSS)} = 5.481 + 0.288 \text{ Ln(XRF-EXT)} - 0.001 (\text{Dist. 2}) \quad 0.25$$

This model indicates that (XRF-EXT) and (Dist. 2) are important determinants of (PbSS). XRF-EXT is also a determinant of lead in (PbSC) obtained near the residence and PbD.WINDOW. Of all the variables examined, only PbSS is predictive of PbD.FLOOR, which in turn is predictive of PbH and PbB. AGE was found to be an important modifier of the PbH → PbB relationship. Other factors such as child's sex, day-care/school attended, socio-economic status, etc. were unrelated to either PbH or PbB. Note that PbSC was not associated with PbD after adjusting for the influence of PbSS. This again suggests that although PbSC is a reservoir of lead with potential for future child exposure, it is the available surface lead (PbSS) which is most associated with other current Pb levels.

The AGE × PbH interaction is illustrated in Figure 2. The expected values, obtained by solving the simultaneous equations for PbH and PbB, are plotted against age. The PbB curve is consistent with previous reports, (Clark *et al.*, 1988) showing that PbB increases during the first 24 months of life, then asymptotes and begins to slowly decline. The PbH curve indicates a continuously increasing amount of Pb recovered from hands as a function of the child's age. This may in part be due to the increase in the surface area of the hand with age or perhaps a change in the amount or type of soil/dust that is contacted. A positive association between PbH and PbB during the first 24 months of life has been reported previously (Bornschein *et al.*, 1985, 1986) and coincides with a period of intense hand-to-mouth and object-to-mouth behavior. The negative association between PbH and PbB in older children suggests several possibilities: 1) hand-to-mouth activity is decreasing and is therefore a less important route for exposure; and/or 2) lead absorption is decreasing so the existing body burden of lead in bone and soft tissue is a proportionately more important determinant of PbB than the Pb currently being ingested via hand-to-mouth activity.

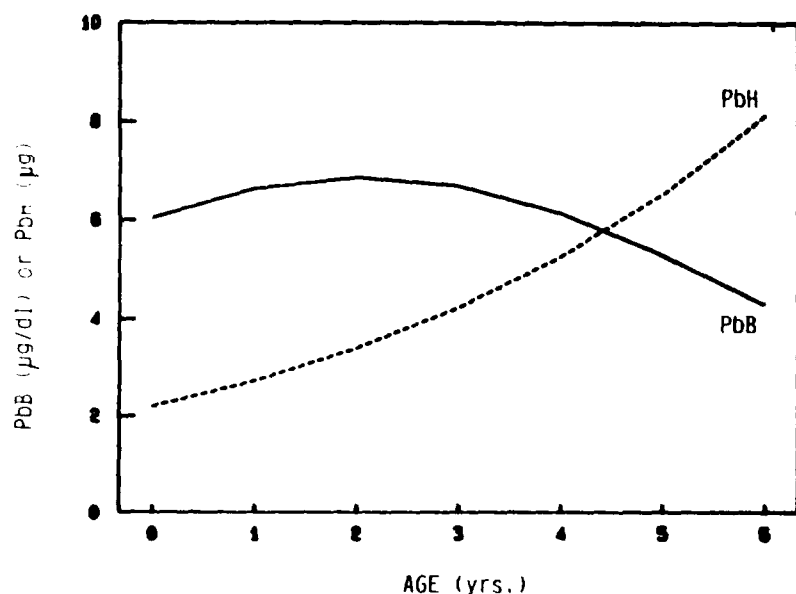


Figure 2. Change in blood lead (PbB) and hand lead (PbH) as a function of age of child. Curves show mean values estimated from the structural equations parameter estimates.

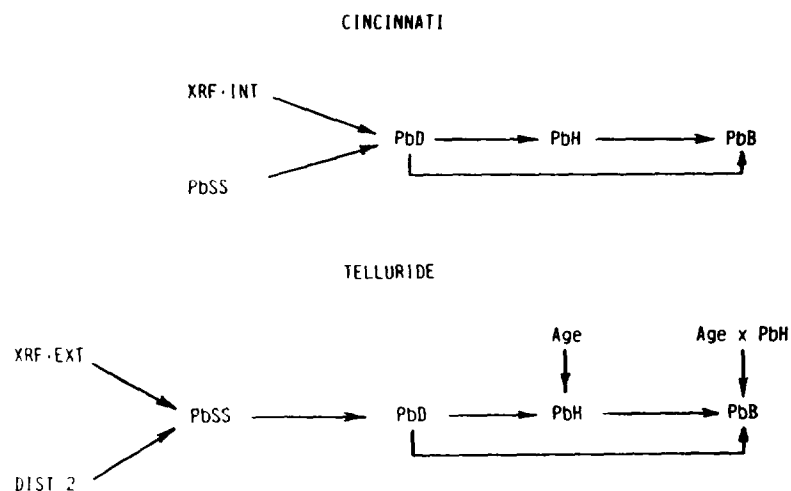


Figure 3. Qualitative summary of the models derived from the Cincinnati and Telluride cohorts. The Cincinnati model was derived for 18 month olds only. The Telluride model is based on 6 to 72 month olds.

Figure 3 reveals the qualitative similarity between the exposure model derived for a cohort of 18-month old children in Cincinnati (Bornschein *et al.*, 1986) and the present model derived for one to six year olds in Telluride. The similarity of the models is striking given the marked differences in the racial, social, and developmental status of the cohorts, the sources of lead in the environment and the high blood lead levels in Cincinnati versus the very low levels seen in Telluride. The $PbSS \rightarrow PbD \rightarrow PbH \rightarrow PbB$ path has been demonstrated in both cohorts. The direct $PbD \rightarrow PbB$ path has also been found in both cohorts and might reflect: 1) measurement error in PbH, 2) exposure due to mouthing of other dust contaminated objects and/or 3) inhalation of airborne Pb-dust. In both locales, evidence was obtained in support of the hypothesized indirect pathway from PbSS to PbB, but no evidence was obtained for a direct path. This suggests that the exposure to lead originates with contaminated soil/dust carried into the home. The need for a child to come into direct contact with soil outside the home does not appear to be a necessary precondition for soil Pb to have an impact on children's PbB.

A comparison of the quantitative estimate of the indirect impact of PbSS on blood lead of 18-month old children in Cincinnati and Telluride is given in Table 5. The blood lead estimates are derived by solving the structural

Table 5. Estimated impact of exterior surface scraping lead (PbSS) on blood lead of 18-month old children.

PbSS	50	100	500	1,000	2,000	Δ
Cincinnati	—	12.50	14.00	14.60	15.30	0.12
Telluride	5.40	6.10	8.30	9.40	—	0.22

$\Delta = \mu\text{g/dL}$ increase in PbB per 100 $\mu\text{g/g}$ increase in PbSS between 500 and 1,000 $\mu\text{g/g}$ PbSS.

equations for various PbSS concentrations. The model for the Cincinnati cohort, which has been discussed in more detail elsewhere (Bornschein *et al.*, 1986), is given below:

Structural Equations

	R^2
$\text{Ln}(\text{PbB}) + 1.276 + 0.152 \text{Ln}(\text{PbH}) + 0.182 \text{Ln}(\text{PbD})$	0.38
$\text{Ln}(\text{PbH}) + -0.966 + 0.444 \text{Ln}(\text{PbD})$	0.22
$\text{Ln}(\text{PbD}) + 4.691 + 0.325 \text{Ln}(\text{XRFHAZ}) + 0.268 \text{Ln}(\text{PbSS})$	0.52

The estimated size of the increase in PbB per unit increase in PbSS is highly dependent on the assumed PbB level. This is because of the non-linear relationship between environmental lead exposure and blood lead. An effect size, has been estimated over the PbSS range of 500 to 1,000 $\mu\text{g/g}$ in order to permit comparison with the recent estimate provided by the American Academy of Pediatrics (Statement on Childhood Lead Poisoning, 1987). The effect size in the Cincinnati and Telluride studies are 0.12 and 0.22 $\mu\text{g/dL}$ per 100 $\mu\text{g/g}$ increase in PbSS, respectively, a factor of 10

lower than suggested in the statement by the American Academy of Pediatrics.

The estimated effect size from 100 to 1,000 $\mu\text{g/g}$ PbSS (Cincinnati + 2.3; Telluride + 3.7 $\mu\text{g/dL}$ per 1,000 $\mu\text{g/g}$) is more consistent with the effect size of 2 $\mu\text{g/dL}$ per 1,000 $\mu\text{g/g}$ estimated by the EPA (Air Quality Criteria for Lead, 1986). The larger effect size seen in the Telluride children is most likely a function of their lower body burden. The reasoning is as follows: there are two major sources of lead in blood; 1) current environmental lead intake and 2) input from tissue and bone lead stores which reflects historical lead intake. In children with low lifetime exposures, the latter factor is relatively small and a given amount of environmental lead intake produces a proportionately larger increment of blood lead than seen in the case of higher lifetime exposures. In the case of children with chronic elevated lead exposure, their higher body lead burden attenuates the impact of environmental lead intake on blood lead.

When comparing the results of the present Telluride blood lead survey with that conducted in 1977, it is important to recognize that there has been a decline in mean population blood lead values over the last two decades.

The average decline per year has been about 1.0 to 1.5 $\mu\text{g/dL}$. Several studies which have documented this decline are summarized in Table 6. In Telluride, in 1977, the average blood lead was found to be 17.5 $\mu\text{g/dL}$. In 1986 we found it to be 6.1 $\mu\text{g/dL}$, a decline of 11.4 $\mu\text{g/dL}$. The average decline per year is 1.27 $\mu\text{g/dL}$, a value very similar to the other studies summarized in Table 6. Thus, Telluride's blood lead levels are following national trends and do not appear to be unduly influenced by the presence of mine tailings.

The low PbB levels observed in the children in Telluride, in the presence of high soil lead concentrations in several "hot spots" in the town, is most likely due to low transfer from the environment to the child and low bioavailability of soil Pb. A major fraction of lead in the soil of Telluride

exists in the form of large particles. A soil size distribution analysis revealed that 82% of the soil lead was found in particles greater than 149 μm (Skogerboe, 1987). These large particles are less readily transported by wind. More importantly, it has been found that the diameter of particles adhering to children's hands is usually less than 10 μm and that it is very uncommon to find particles as large as 100 μm adhering to a child's hand (Duggan and Inskip, 1985; Duggan *et al.*, 1985). Thus, the large lead-containing particles in Telluride soil are not readily consumed during the course of normal hand-to-mouth activity. Furthermore, a significant fraction of the lead in Telluride soil is of geologic origin, *i.e.* lead sulphide imbedded in a quartz matrix. Lead in this form is much less likely to be dissolved and absorbed in the gut than other forms of lead such as those derived from paint or automotive emissions (Bartrop, 1975; Chaney and Mielke, 1986). Finally, much of the lead-containing soil found around children's residences was well covered by grass, thereby making it less accessible to direct contact and less available for tracking into the home. All of these factors work to reduce the potential hazard of the environmental lead and are consistent with the observed low blood lead levels in the community.

Conclusions

The results of the analysis of data from Telluride confirm the exposure model developed in the Cincinnati cohort. The indirect pathway, PbSS \rightarrow PbD \rightarrow PbH \rightarrow PbB has been demonstrated in the present study and is qualitatively and quantitatively similar to that obtained in a distinctly different cohort and exposure situation in Cincinnati. Although lead contaminated hand dust is an important component of the exposure pathway, its impact on blood lead is age dependent and largely disappears beyond four years of age. This study shows that although the effect size is small, it is possible to demonstrate the impact of PbSS on PbB at concentrations less than 500 $\mu\text{g/g}$. The magnitude of the effect is in the range of 2–4 $\mu\text{g/dL}$ per 1,000 $\mu\text{g/g}$ Pb in surface scrapings, a value consistent with the EPA estimate of 2 $\mu\text{g/dL}$ per 1,000 $\mu\text{g/g}$ Pb for soil. Finally in any assessment of risk due to soil lead, consideration must be given to accessibility of the soil, particle size, chemical speciation and bioavailability of the lead.

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Table 6. Time trends for blood lead levels.

Study	Sample	Rate of decline ($\mu\text{g/dL/yr}$)	PbB [yr] ($\mu\text{g/dL}$)	References
Boston	N = 11,837 cord blood	0.89	7.5 [1979] 5.7 [1981]	Rabinowitz and Needleman, 1982
NHANESII	N = 9,933	1.20	14.6 [1976]	Mahaffey
National	6 month–74 year		9.2 [1980]	<i>et al.</i> , 1977
Survey	N = 2,372	1.40	15.0 [1976]	
	6 month–60 month		8.7 [1980]	
New York	N = 115,000 children	1.50	24.0 [1970] 5.1 [1976]	Billick <i>et al.</i> , 1979
Chicago	—	1.33	30.0 [1970] 18.0 [1979]	Billick <i>et al.</i> , 1979
Cincinnati	N = 215 24 month	0.97	19.6 [1982] 15.7 [1986]	Unpublished
Telluride	N = about 90 children in each survey	1.27	17.5 [1977] 6.1 [1986]	This study and 1977 survey

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Modelling the Blood Lead - Soil Lead Relationship*

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Abstract

The relationship between blood lead and lead uptake from the environment has been described by cross-sectional models, by causal models based on longitudinal data, and by predictive models based on hypotheses about uptake and biokinetics in children. The empirical blood lead versus soil linear slope in many studies is about 2 µg/dL per 1,000 µg/g soil lead. Uptake/biokinetic models are currently being validated and give estimates of mean blood lead in children. Biological and statistical factors may cause nonlinearity of the relationship at blood lead levels above 2 µg/dL.

Introduction

Elevated lead levels in surface soil and environmental dust cause elevated blood lead concentrations in many young children who spend extended periods of time living or playing near the contaminated soil.

The mechanisms are understood in general. Surface dust may be ingested by normal hand-to-mouth activity, especially in one- to three-year-old children (Duggan *et al.*, 1985). Some may consume excess large quantities of soil and other non-food items, a condition known as *pica*. Contaminated soil may become accessible by weathering, or by humans or animals digging up the soil. Soil particles can become airborne and be inhaled, can contribute to internal household dust by atmospheric entry or can be tracked into the house on shoes, clothing and pets. Further work on the quantification of these processes is needed.

*This paper represents the views of the authors and does not represent the view of Battelle Memorial Institute or the United States Environmental Protection Agency.